

**What is claimed is:**

1. An antagonist that inhibits or an agonist that activates an activity a polypeptide selected from the group consisting of: a polypeptide comprising an amino acid sequence which is at least 90% identical to the amino acid sequence of SEQ ID NO:2 or 4,  
5 and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:2 or 4, wherein said activity is selected from the group consisting of:
    - uncompetitive inhibition by Apo-ACP versus NADH ( $K_{iapp}$ );
    - competitive inhibition by Apo-ACP versus crotonoyl CoA;
    - induction of negative cooperativity with respect to CCA binding;
  - 10 use of NADH and NADPH as substrates by Fab I;
    - binding of NADH and NADPH by FabI;
    - oxidation of NADH and NADPH by FabI;
    - ratio of  $K_{mapp}$  for NADH as compared to NADPH;
    - use of NADH and crotonoyl CoA as substrates by Fab I in a sequential  
15 kinetic mechanism;
    - sequential binding of NADH and crotonoyl CoA by Fab I;
    - increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length;
    - feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
  - 20 competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
    - competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;
    - binding of multiple palmitoyl CoA molecules to Fab I;
    - negative cooperativity in the binding of CCA;
  - 25 formation of an dimeric quaternary structure;
    - formation of an tetrameric quaternary structure;
    - formation of an oligomeric quaternary structure;
    - binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and
  - 30 NADH binding to Fab I prior to or simultaneous with ACP binding.
2. A method for the treatment of an individual having need to inhibit or activate Fab I polypeptide comprising the steps of: administering to the individual a antibacterially

effective amount of an antagonist that inhibits or an agonist that activates an activity of a polypeptide selected from the group consisting of: a polypeptide comprising an amino acid sequence which is at least 90% identical to the amino acid sequence of SEQ ID NO:2 or 4, and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:2 or 4.

5       wherein said activity is selected from the group consisting of:

- uncompetitive inhibition by Apo-ACP versus NADH ( $K_{iapp}$ );
- competitive inhibition by Apo-ACP versus crotonoyl CoA;
- induction of negative cooperativity with respect to CCA binding;
- use of NADH and NADPH as substrates by Fab I;

10      binding of NADH and NADPH by FabI;

- oxidation of NADH and NADPH by FabI;
- ratio of  $K_{mapp}$  for NADH as compared to NADPH;
- use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;

15      sequential binding of NADH and crotonoyl CoA by Fab I;

- increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length;
- feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
- competitive inhibition by palmitoyl CoA versus crotonoyl CoA;

20      competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;

- binding of multiple palmitoyl CoA molecules to Fab I;
- negative cooperativity in the binding of CCA;
- formation of an dimeric quaternary structure;

25      formation of an tetrameric quaternary structure;

- formation of an oligomeric quaternary structure;
- binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and

30      NADH binding to Fab I prior to or simultaneous with ACP binding.

3.       A method for the treatment of an individual infected with a bacteria comprising the steps of administering to the individual a antibacterially effective amount of an antagonist that inhibits or an agonist that activates an activity of a polypeptide selected from

the group consisting of: a polypeptide comprising an amino acid sequence which is at least 90% identical to the amino acid sequence of SEQ ID NO:2 or 4, and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:2 or 4, wherein said activity is selected from the group consisting of:

- 5           uncompetitive inhibition by Apo-ACP versus NADH (Kitapp);  
          competitive inhibition by Apo-ACP versus crotonoyl CoA;  
          induction of negative cooperativity with respect to CCA binding;  
          use of NADH and NADPH as substrates by Fab I;  
          binding of NADH and NADPH by FabI;  
10          oxidation of NADH and NADPH by FabI;  
          ratio of Kmapp for NADH as compared to NADPH;  
          use of NADH and crotonoyl CoA as substrates by Fab I in a sequential  
          kinetic mechanism;  
          sequential binding of NADH and crotonoyl CoA by Fab I;  
15          increasing inhibition of FabI by saturated fatty acyl CoA's of increasing  
          chain length; feedback regulatory mechanism of Fab I by saturated fatty  
          acyl CoA's;  
          competitive inhibition by palmitoyl CoA versus crotonoyl CoA;  
          competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation  
20          through binding of multiple palmitoyl CoA molecules to Fab I;  
          binding of multiple palmitoyl CoA molecules to Fab I;  
          negative cooperativity in the binding of CCA;  
          formation of an dimeric quaternary structure;  
          formation of an tetrameric quaternary structure;  
25          formation of an oligomeric quaternary structure;  
          binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl  
          coA; and  
          NADH binding to Fab I prior to or simultaneous with ACP binding.
- 4.         The method of claim 3 wherein said bacteria is selected from the group  
30         consisting of a member of the genus *Staphylococcus*, *Staphylococcus aureus*, a member of the  
          genus *Streptococcus*, and *Streptococcus pneumoniae*.

5. A method for the treatment of an individual having need to inhibit or activate Fab I polypeptide comprising the steps of administering to the individual a antibacterially effective amount of an antagonist that inhibits or an agonist that activates an activity of Fab I selected from the group consisting of:

- 5 uncompetitive inhibition by Apo-ACP versus NADH (K<sub>iapp</sub>);
- competitive inhibition by Apo-ACP versus crotonoyl CoA;
- induction of negative cooperativity with respect to CCA binding;
- use of NADH and NADPH as substrates by Fab I;
- binding of NADH and NADPH by FabI;
- 10 oxidation of NADH and NADPH by FabI;
- ratio of K<sub>mapp</sub> for NADH as compared to NADPH;
- use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;
- sequential binding of NADH and crotonoyl CoA by Fab I;
- 15 increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
- competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
- competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation
- 20 through binding of multiple palmitoyl CoA molecules to Fab I;
- binding of multiple palmitoyl CoA molecules to Fab I;
- negative cooperativity in the binding of CCA;
- formation of an dimeric quaternary structure;
- formation of an tetrameric quaternary structure;
- 25 formation of an oligomeric quaternary structure;
- binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and
- NADH binding to Fab I prior to or simultaneous with ACP binding.

6. A method for the treatment of an individual infected with a bacteria comprising the steps of administering to the individual a antibacterially effective amount of an antagonist that inhibits or an agonist that activates that activates an activity of Fab I selected from the group consisting of:

- uncompetitive inhibition by Apo-ACP versus NADH (Ki(app));  
competitive inhibition by Apo-ACP versus crotonoyl CoA;  
induction of negative cooperativity with respect to CCA binding;  
use of NADH and NADPH as substrates by Fab I;  
5 binding of NADH and NADPH by FabI;  
oxidation of NADH and NADPH by FabI;  
ratio of Kmapp for NADH as compared to NADPH;  
use of NADH and crotonoyl CoA as substrates by Fab I in a sequential  
kinetic mechanism;  
10 sequential binding of NADH and crotonoyl CoA by Fab I;  
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing  
chain length; feedback regulatory mechanism of Fab I by saturated fatty  
acyl CoA's;  
competitive inhibition by palmitoyl CoA versus crotonoyl CoA;  
15 competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation  
through binding of multiple palmitoyl CoA molecules to Fab I;  
binding of multiple palmitoyl CoA molecules to Fab I;  
negative cooperativity in the binding of CCA;  
formation of an dimeric quaternary structure;  
20 formation of an tetrameric quaternary structure;  
formation of an oligomeric quaternary structure;  
binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl  
coA; and  
NADH binding to Fab I prior to or simultaneous with ACP binding.  
25 7. The method of claim 6 wherein said bacteria is selected from the group  
consisting of: a member of the genus *Staphylococcus*, *Staphylococcus aureus*, a member of  
the genus *Streptococcus*, and *Streptococcus pneumoniae*.  
8. A method for the treatment of an individual infected by *Streptococcus*  
30 *pneumoniae* comprising the steps of administering to the individual a antibacterially effective  
amount of an antagonist that inhibits or anagonist that activates an activity of *Streptococcus*  
*pneumoniae* Fab I selected from the group consisting of:  
uncompetitive inhibition by Apo-ACP versus NADH (Ki(app));

- competitive inhibition by Apo-ACP versus crotonoyl CoA;  
induction of negative cooperativity with respect to CCA binding;  
use of NADH and NADPH as substrates by Fab I;  
binding of NADH and NADPH by FabI;  
5 oxidation of NADH and NADPH by FabI;  
ratio of Kmapp for NADH as compared to NADPH;  
use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;  
sequential binding of NADH and crotonoyl CoA by Fab I;  
10 increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;  
competitive inhibition by palmitoyl CoA versus crotonoyl CoA;  
competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation  
15 through binding of multiple palmitoyl CoA molecules to Fab I;  
binding of multiple palmitoyl CoA molecules to Fab I;  
negative cooperativity in the binding of CCA;  
formation of an dimeric quaternary structure;  
formation of an tetrameric quaternary structure;  
20 formation of an oligomeric quaternary structure;  
binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and  
NADH binding to Fab I prior to or simultaneous with ACP binding.
9. An antagonist that inhibits an activity of a polypeptide selected from the  
25 group consisting of: a polypeptide comprising an amino acid sequence which is at least 90% identical to the amino acid sequence of SEQ ID NO:2 or 4, and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:2 or 4, , wherein said activity is selected from the group consisting of:
- uncompetitive inhibition by Apo-ACP versus NADH ( $K_i$ (app));  
30 competitive inhibition by Apo-ACP versus crotonoyl CoA;  
induction of negative cooperativity with respect to CCA binding;  
use of NADH and NADPH as substrates by Fab I;

- binding of NADH and NADPH by FabI;  
oxidation of NADH and NADPH by FabI;  
ratio of Kmapp for NADH as compared to NADPH;  
use of NADH and crotonoyl CoA as substrates by Fab I in a sequential  
kinetic mechanism;
- sequential binding of NADH and crotonoyl CoA by Fab I;  
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing  
chain length; feedback regulatory mechanism of Fab I by saturated fatty  
acyl CoA's;
- competitive inhibition by palmitoyl CoA versus crotonoyl CoA;  
competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation  
through binding of multiple palmitoyl CoA molecules to Fab I;  
binding of multiple palmitoyl CoA molecules to Fab I;  
negative cooperativity in the binding of CCA;
- formation of an dimeric quaternary structure;  
formation of an tetrameric quaternary structure;  
formation of an oligomeric quaternary structure;  
binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl  
coA; and
- NADH binding to Fab I prior to or simultaneous with ACP binding.
10. A method for the treatment of an individual having need to inhibit Fab I polypeptide comprising the steps of administering to the individual a antibacterially effective amount of an antagonist that inhibits an activity of a polypeptide selected from the group consisting of a polypeptide comprising an amino acid sequence which is at least 90% identical to the amino acid sequence of SEQ ID NO:2 or 4, and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:2 or 4, wherein said activity is selected from the group consisting of:
- uncompetitive inhibition by Apo-ACP versus NADH ( $K_i(app)$ );  
competitive inhibition by Apo-ACP versus crotonoyl CoA;
- induction of negative cooperativity with respect to CCA binding;  
use of NADH and NADPH as substrates by Fab I;  
binding of NADH and NADPH by FabI;

- oxidation of NADH and NADPH by FabI;  
ratio of  $K_m$  for NADH as compared to NADPH;  
use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;
- 5 sequential binding of NADH and crotonoyl CoA by Fab I;  
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;  
competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
- 10 competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;  
binding of multiple palmitoyl CoA molecules to Fab I;  
negative cooperativity in the binding of CCA;  
formation of an dimeric quaternary structure;
- 15 formation of an tetrameric quaternary structure;  
formation of an oligomeric quaternary structure;  
binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and  
NADH binding to Fab I prior to or simultaneous with ACP binding.
- 20 11. A method for inhibiting an activity of Fab I polypeptide comprising the steps of contacting a composition comprising said polypeptide with an effective amount of an antagonist that inhibits an activity of Fab I, wherein said activity is selected from the group consisting of:  
uncompetitive inhibition by Apo-ACP versus NADH ( $K_i$ (app));  
25 competitive inhibition by Apo-ACP versus crotonoyl CoA;  
induction of negative cooperativity with respect to CCA binding;  
use of NADH and NADPH as substrates by Fab I;  
binding of NADH and NADPH by FabI;  
oxidation of NADH and NADPH by FabI;
- 30 ratio of  $K_m$  for NADH as compared to NADPH;  
use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;

- sequential binding of NADH and crotonoyl CoA by Fab I;  
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
- 5 competitive inhibition by palmitoyl CoA versus crotonoyl CoA;  
competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;  
binding of multiple palmitoyl CoA molecules to Fab I;  
negative cooperativity in the binding of CCA;
- 10 formation of an dimeric quaternary structure;  
formation of an tetrameric quaternary structure;  
formation of an oligomeric quaternary structure;  
binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and
- 15 NADH binding to Fab I prior to or simultaneous with ACP binding.
12. A method for inhibiting an activity of Fab I, wherein said activity is selected from the group consisting of:
- uncompetitive inhibition by Apo-ACP versus NADH ( $K_{iapp}$ );  
competitive inhibition by Apo-ACP versus crotonoyl CoA;
- 20 induction of negative cooperativity with respect to CCA binding;  
use of NADH and NADPH as substrates by Fab I;  
binding of NADH and NADPH by FabI;  
oxidation of NADH and NADPH by FabI;  
ratio of  $K_{mapp}$  for NADH as compared to NADPH;
- 25 use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;  
sequential binding of NADH and crotonoyl CoA by Fab I;  
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
- 30 competitive inhibition by palmitoyl CoA versus crotonoyl CoA;

- competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;  
binding of multiple palmitoyl CoA molecules to Fab I;  
negative cooperativity in the binding of CCA;  
5 formation of an dimeric quaternary structure;  
formation of an tetrameric quaternary structure;  
formation of an oligomeric quaternary structure;  
binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and  
10 NADH binding to Fab I prior to or simultaneous with ACP binding.

13. The method of claim 12 wherein said bacteria is selected from the group consisting of: a member of the genus *Staphylococcus*, *Staphylococcus aureus*, a member of the genus *Streptococcus*, and *Streptococcus pneumoniae*.

14. A method for inhibiting a growth of bacteria comprising the steps of contacting a composition comprising bacteria with an antibacterially effective amount of an antagonist that inhibits an activity of Fab I, wherein said activity is selected from the group consisting of:

- uncompetitive inhibition by Apo-ACP versus NADH (Ki(app));  
competitive inhibition by Apo-ACP versus crotonoyl CoA;  
20 induction of negative cooperativity with respect to CCA binding;  
use of NADH and NADPH as substrates by Fab I;  
binding of NADH and NADPH by FabI;  
oxidation of NADH and NADPH by FabI;  
ratio of Kmapp for NADH as compared to NADPH;  
25 use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;  
sequential binding of NADH and crotonoyl CoA by Fab I;  
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;  
30 competitive inhibition by palmitoyl CoA versus crotonoyl CoA;

competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;  
binding of multiple palmitoyl CoA molecules to Fab I;  
negative cooperativity in the binding of CCA;  
5 formation of an dimeric quaternary structure;  
formation of an tetrameric quaternary structure;  
formation of an oligomeric quaternary structure;  
binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and  
10 NADH binding to Fab I prior to or simultaneous with ACP binding.

15. The method of claim 14 wherein said bacteria is selected from the group consisting of: a member of the genus *Staphylococcus*, *Staphylococcus aureus*, a member of the genus *Streptococcus*, and *Streptococcus pneumoniae*.

16. A method for inhibiting a Fab I polypeptide comprising the steps of contacting a composition comprising bacteria with an antibacterially effective amount of an antagonist that inhibits an activity of Fab I, wherein said activity is selected from the group consisting of:  
uncompetitive inhibition by Apo-ACP versus NADH ( $K_i(\text{app})$ );  
competitive inhibition by Apo-ACP versus crotonoyl CoA;  
20 induction of negative cooperativity with respect to CCA binding;  
use of NADH and NADPH as substrates by Fab I;  
binding of NADH and NADPH by FabI;  
oxidation of NADH and NADPH by FabI;  
ratio of  $K_{\text{mapp}}$  for NADH as compared to NADPH;  
25 use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;  
sequential binding of NADH and crotonoyl CoA by Fab I;  
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;  
30 competitive inhibition by palmitoyl CoA versus crotonoyl CoA;

- competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I; binding of multiple palmitoyl CoA molecules to Fab I; negative cooperativity in the binding of CCA;
- 5 formation of an dimeric quaternary structure; formation of an tetrameric quaternary structure; formation of an oligomeric quaternary structure; binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and
- 10 NADH binding to Fab I prior to or simultaneous with ACP binding.

17. The method of claim 16 wherein said bacteria is selected from the group consisting of: a member of the genus *Staphylococcus*, *Staphylococcus aureus*, a member of the genus *Streptococcus*, and *Streptococcus pneumoniae*.